

# The lifespan trajectory of neural oscillatory activity in the motor system

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## ABSTRACT

Numerous studies connect beta oscillations in the motor cortices to volitional movement, and beta is known to be aberrant in multiple movement disorders. However, the dynamic interplay between these beta oscillations, motor performance, and spontaneous beta power (e.g., during rest) in the motor cortices remains unknown. This study utilized magnetoencephalography (MEG) to investigate these three parameters and their lifespan trajectory in 57 healthy participants aged 9–75 years old. Movement-related beta activity was imaged using a beamforming approach, and voxel time series data were extracted from the peak voxels in the primary motor cortices. Our results indicated that spontaneous beta power during rest followed a quadratic lifespan trajectory, while movement-related beta oscillations linearly increased with age. Follow-on analyses showed that spontaneous beta power and the beta minima during movement, together, significantly predicted task performance above and beyond the effects of age. These data are the first to show lifespan trajectories among measures of beta activity in the motor cortices, and suggest that the healthy brain compensates for age-related increases in spontaneous beta activity by increasing the strength of beta oscillations within the motor cortices which, when successful, enables normal motor performance into later life.

## 1. Introduction

Transient human movement is served by a specific pattern of neural oscillatory activity, particularly in the beta band (14–30 Hz). Briefly, prior to and during movement, there is a strong decrease in beta activity relative to baseline levels, known as the peri-movement beta event-related desynchronization (ERD), which begins about 1.0 s before movement onset and dissipates shortly after movement concludes (Cheyne et al., 2006; Engel and Fries, 2010; Gaetz et al., 2010; Heinrichs-Graham and Wilson, 2015, 2016; Heinrichs-Graham et al., 2014b; Jurkiewicz et al., 2006; Pfurtscheller and Lopes da Silva, 1999; Wilson et al., 2014, 2010, 2011). This response has been reliably associated with movement planning and execution (Doyle et al., 2005; Grent-t-Jong et al., 2014; Heinrichs-Graham et al., 2016; Heinrichs-Graham and Wilson, 2015; Kaiser et al., 2001; Tzagarakis et al., 2010). Following the beta ERD, there is a strong resynchronization (above

baseline levels), termed the post-movement beta rebound (PMBR), which extends from approximately 0.8–2.5 s after movement has stopped (Cheyne et al., 2006; Gaetz et al., 2010; Heinrichs-Graham et al., 2014b; Jurkiewicz et al., 2006; Pfurtscheller and Lopes da Silva, 1999; Wilson et al., 2010, 2011). During simple movements, these beta-band oscillations reliably peak in the precentral gyri bilaterally with stronger activity contralateral to movement, while more complex movements (and some simple movements) also induce activity in the supplementary motor area and bilateral premotor cortices, postcentral gyri, parietal cortices, and cerebellum (Cheyne et al., 2006, 2008; Fry et al., 2016; Gaetz and Cheyne, 2006; Gaetz et al., 2010; Heinrichs-Graham et al., 2016; Heinrichs-Graham and Wilson, 2015, 2016; Heinrichs-Graham et al., 2014b; Jurkiewicz et al., 2006; Muthukumaraswamy, 2010; Wilson et al., 2010).

Prior studies have shown that these movement-related oscillatory patterns change as a function of age (Gaetz et al., 2010; Rossiter et al.,

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2014; Wilson et al., 2010). For example, a magnetoencephalography (MEG) study of simple movements showed that the beta ERD becomes stronger (i.e., more negative relative to baseline) and the PMBR linearly increases from childhood to early adulthood, with young children (aged 4–6 years) exhibiting little-to-no PMBR response (Gaetz et al., 2010). Another MEG study showed a similar linear increase in beta ERD from early to late adulthood (Rossiter et al., 2014). Interestingly, this study also found a significant age-related linear increase in spontaneous (i.e., no task) beta activity in the motor cortices (Rossiter et al., 2014). Recently, our laboratory utilized MEG and a motor sequencing task to directly probe the relationship between spontaneous beta activity and motor-related oscillations in the context of healthy aging (Heinrichs-Graham and Wilson, 2016). Consistent with previous findings, we found that older adults exhibited an almost threefold increase in spontaneous beta power in the primary motor cortices, as well as significantly stronger beta ERD in the same regions compared to younger adults. Taken together, these studies provide substantial evidence that there are major neurophysiological changes that occur in the motor cortices throughout the lifespan.

Importantly, the aforementioned study from our laboratory (Heinrichs-Graham and Wilson, 2016) also found a direct linear relationship between spontaneous beta power and peri-movement beta ERD power, such that with greater spontaneous (resting) beta, there was greater baseline-relative beta suppression (i.e., ERD) during movement. In addition, we found a significant relationship between baseline-relative beta ERD and movement duration, such that the greater the decrease in beta power relative to baseline levels, the longer the movement duration (Heinrichs-Graham and Wilson, 2016). This pattern of results suggests that spontaneous beta activity in the primary motor cortex and movement-related beta ERD power are directly related, and that the relationship between these two measures affects motor performance (see Fig. 1). This significant link between spontaneous and motor-related beta oscillatory activity also corroborated an earlier MEG study (Wilson et al., 2014), which investigated the impact of time-of-day on motor-related oscillatory activity. This study found a linear increase in peri-movement beta ERD power (i.e., more negative relative to baseline), coupled with a roughly proportional increase in spontaneous beta power, as a function of time of day (Wilson et al., 2014). While not directly investigated in this study, this pattern of results clearly suggested that the two neurophysiological measures were linked, as the stronger peri-movement beta ERD during movement appeared to be offsetting the increased spontaneous beta levels during both the baseline period and a separately-acquired resting state recording (Wilson et al., 2014).

While these and other studies have independently suggested that there is a unique change in beta oscillatory activity as a function of age and that these oscillatory measures are directly related, no study to date has looked at the nature of this relationship across the lifespan. Basically, studies have shown differences in movement-related beta

activity from youth to adulthood (Gaetz et al., 2010), while others have shown differences in beta activity from younger to older adulthood (Heinrichs-Graham and Wilson, 2016; Rossiter et al., 2014), but critically missing is the developmental trajectory of these measures from youth to late adulthood. Such information could provide invaluable data on functional brain maturation, and serve as a baseline by which pathology could be assessed. Furthermore, examining these responses across the lifespan would provide a powerful testbed for determining whether spontaneous beta levels are directly related to the strength of the beta ERD. In the current study, we used a complex motor sequence paradigm to study the relationship between spontaneous beta activity and movement-related beta oscillations in the motor cortices from preadolescence through late adulthood. We first sought to determine how spontaneous activity in the motor cortex and movement-related beta oscillations change as a function of age. Secondly, we aimed to examine the relationship between spontaneous beta activity and movement-related oscillations in the context of development and aging (Fig. 1). We hypothesized that there would be unique developmental trajectories for both spontaneous and movement-related beta oscillatory responses. Further, we hypothesized that there would be a tight link between these responses, and that this relationship would mediate motor performance across the lifespan.

## 2. Material and methods

### 2.1. Subject selection

A total of 57 males were enrolled in the study. We focused on males in this study due to several recent reports of sex differences in the aging brain (Scheinost et al., 2015; Shaw et al., 2016). All participants were recruited from the local community. Data from the adults were included in another recent publication (Heinrichs-Graham and Wilson, 2016); however, these data were fully re-analyzed as described below. Thus, all reported results, with the exception of the adult behavioral data, are unique to this publication. Exclusionary criteria included inability to perform the task, any medical illness affecting CNS function, neurological or psychiatric disorder, history of head trauma, current substance abuse, any medication known to affect CNS function, and the MEG Laboratory's standard exclusion criteria (e.g., dental braces, metal implants, battery operated implants, and/or any type of ferromagnetic implanted material). After complete description of the study was given to participants, written informed consent was obtained from the adult participants and parents of the youth participants, and informed assent was obtained from the youth participants, following the guidelines of the University of Nebraska Medical Center's Institutional Review Board which approved the study protocol. Six additional youth were recruited, but excluded from analysis due to our standard MEG exclusionary criteria (e.g., movement artifacts, inability to perform the task).

### 2.2. Experimental paradigm and stimuli

During MEG recording, participants were seated in a nonmagnetic chair within the magnetically-shielded room, and each participant rested their right hand on a custom-made five-finger button pad. This response pad was connected such that each button sent a unique signal (i.e., TTL pulse/trigger code) to the MEG system acquisition computer, and thus behavioral responses were temporally synced with the MEG data. This allowed accuracy, reaction times (i.e., time between the cue to move and first button press), and movement durations (i.e., how long it took to complete the tapping sequence) in ms to be computed offline. Each participant first completed a motor sequencing task, during which they were instructed to complete a series of finger-tapping sequences as quickly and accurately as possible. During the motor sequencing task, participants fixated on a crosshair presented centrally. After a sufficient baseline period of 3.75 s, a series of three numbers, each corresponding to a finger on the hand, was presented on the screen in black for 0.5 s.

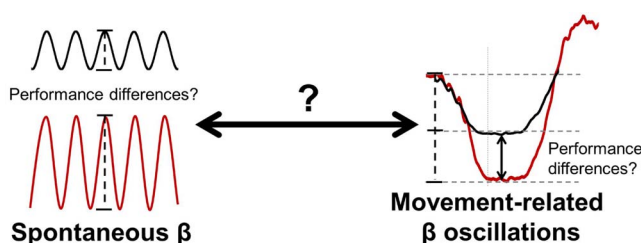


Fig. 1. Proposed relationship between spontaneous beta power and movement-related beta oscillations in the primary motor cortex. The central goals of this study were to identify the dynamic link between spontaneous (i.e., no task) beta power (left) and movement-related beta oscillations (right), and to determine their combined impact on motor performance throughout the lifespan. Previous studies have shown that spontaneous beta levels in the motor cortices sharply increase in later life (Heinrichs-Graham and Wilson, 2016; Rossiter et al., 2014).

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